| Set | Items | Description |
|---|---|---|
| S1 | 96552 | TUMOR(W)NECROSIS(W)FACTOR OR TNF |
| S2 | 102699 | TRANSFECT? |
| S3 | 1198238 | EXPRESS? |
| S4 | 28243 | S1 AND (S2 OR S3) |
| S5 | 599091 | VIVO |
| S6 | 3586 | S4 AND S5 |
| S7 | 909173 | RADIATION |
| S8 | 59 | S6 AND S7 |
| S9 | 33 | RD (unique items) |
| S10 | 526 | S6 AND INJECT? |
| S11 | 237 | RD (unique items) |
| S12 | 1391 | S6 AND (GENE OR VECTOR) |
| S13 | 1171 | S12 NOT S10 |
| S14 | 40931 | NUDE |
| S15 | 43 | S13 AND S14 |
| S16 | 20 | RD (unique items) |
| S17 | 21474 | GENE(W)THERAPY |
| S18 | 37306 | ADENOVIR? |
| S19 | 85762 | HERPES OR HSV |
| S20 | 1126660 | TUMOR? ? |
| S21 | 439 | S17 AND S18 AND S20 |
| S22 | 273 | RD (unique items) |
| S23 | 4251 | S18 AND S20 |
| S24 | 4701519 | PY=1994:1996 |
| S25 | 2997 | S23 NOT S24 |
| S26 | 1306223 | GENE OR VECTOR |
| S27 | 1060 | S25 AND S26 |
| S28 | 711355 | INJECT? |
| S29 | 38 | S27 AND S28 |
| S30 | 25 | RD (unique items) |
| S31 | 577 | INTRATUMORAL(W)INJECTION |
| S32 | 11 | S31 AND S18 |
| S33 | 5 | RD (unique items) |
| S34 | 35 | S31 AND S19 |
| S35 | 17 | RD (unique items) |
| S36 | 75 | S26 AND S31 |
| S37 | 12 | S36 NOT S24 |
| S38 | 4 | RD (unique items) |
| S39 | 3743 | S20 AND S26 AND S28 |
| S40 | 1805 | S39 NOT S24 |
| S41 | 481 | S40 AND S14 |
| S42 | 243 | RD (unique items) |

?t s11/9/115,231,236

11/9/115 (Item 115 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


9628772 BIOSIS Number: 94133772
IN-*VIVO* ACTIVITY OF *TUMOR* *NECROSIS* *FACTOR* *TNF* MUTANTS SECRETORY BUT NOT MEMBRANE-BOUND *TNF* MEDIATES THE REGRESSION OF RETROVIRALLY TRANSDUCED MURINE TUMOR

KARP S E; HWU P; FARBER A; RESTIFO N P; KRIEGLER M; MULE J J; ROSENBERG S A
NATL. INST. HEALTH, BUILD. 10, ROOM 2B46, BETHESDA, MD. 20892.

J IMMUNOL 149 (6). 1992. 2076-2081. CODEN: JOIMA
Full Journal Title: Journal of Immunology
Language: ENGLISH

We have previously demonstrated that murine tumor cells transduced with a retrovirus containing the cDNA encoding wild-type human *TNF* regress in *vivo* when *injected* into immunocompetent mice; this regression is T cell mediated. To determine whether membrane-associated or secreted *TNF* was responsible for tumor regression, we transduced a cloned murine fibrosarcoma 205 F4 with retroviruses encoding modified human *TNF* genes. The cloned tumor lines of one retroviral transduction *expressed* only membrane bound 26-kDa *TNF*. This *TNF* could not be cleaved or secreted, but was present on the cell surface. A second retrovirus caused the *expression* of only secretory 17-kDa *TNF*, as the transmembrane domain of the cDNA was deleted. The *TNF* produced by tumor cells transduced with either retroviral vector was functional in vitro as direct lysis of the *TNF*-sensitive target L929 by transduced tumor cells was demonstrated. The *TNF* present on 26-kDa *expressing* tumors was membrane bound as supernatants from cultured 17-kDa *TNF* *expressing* tumor cells but not 26-kDa *TNF* *expressing* tumors mediated the lysis of L929 cells. Both tumors were *injected* s.c. into syngeneic mice and tumor growth was measured serially. In repeated experiments, 26-kDa *TNF* *expressing* tumors grew progressively in all mice. In contrast, 17-kDa *TNF* *expressing* tumors grew for 10 days and then regressed with all animals free of tumor at 28 days. Tumor regression was abrogated by in *vivo* *injection* of anti-*TNF* antibody. Similar results were obtained in a second tumor model, 203 E4. Thus regression of *TNF* transduced tumors in *vivo* requires secretion of *TNF*, as membrane-bound *TNF* is insufficient to elicit the host response.

Descriptors/Keywords: HUMAN COMPLEMENTARY DNA FIBROSARCOMA T CELL ANTITUMOR GENE THERAPY HOST IMMUNE RESPONSE

Concept Codes:

| | |
|---|---|
| *02506 | Cytology and Cytochemistry-Animal |
| *02508 | Cytology and Cytochemistry-Human |
| *03506 | Genetics and Cytogenetics-Animal |
| *03508 | Genetics and Cytogenetics-Human |
| *15004 | Blood, Blood-Forming Organs and Body Fluids-Blood Cell Studies |
| *15008 | Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System |
| *17002 | Endocrine System-General |
| *24003 | Neoplasms and Neoplastic Agents-Immunology |
| *24006 | Neoplasms and Neoplastic Agents-Biochemistry |
| *24007 | Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis |
| *31500 | Genetics of Bacteria and Viruses |
| *34502 | Immunology and Immunochemistry-General; Methods |
| *36006 | Medical and Clinical Microbiology-Virology |
| 10062 | Biochemical Studies-Nucleic Acids, Purines and Pyrimidines |
| 10064 | Biochemical Studies-Proteins, Peptides and Amino Acids |

Biosystematic Codes:

| | |
|---|---|
| 02240 | Retroviridae-Unspecified (1979- ) |
| 86215 | Hominidae |
| 86375 | Muridae |

Super Taxa:
Microorganisms; Viruses; Animals; Chordates; Vertebrates; Mammals; Primates; Humans; Nonhuman Vertebrates; Nonhuman Mammals; Rodents

11/9/231 (Item 4 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

180899 DBA Accession No.: 95-08919
Cytokines and clinical gene therapy - cytokine-mediated gene therapy; a review
AUTHOR: Schmidt-Wolf G; +Schmidt-Wolf I G H
CORPORATE AFFILIATE: Univ.Berlin-Free
CORPORATE SOURCE: Abteilung Innere Medizin m.S., Haematologie und Onkologie, Universitaetsklinikum Rudolf Virchow, Spandauer Damm 130, D-14050 Berlin, Germany.
JOURNAL: Eur.J.Immunol. (25, 4, 1137-40) 1995
ISSN: 0014-2980 CODEN: EJIMAF
LANGUAGE: English
ABSTRACT: An alternative means of cytokine delivery is the *transfection* of the cytokine gene into tumor or carrier cells that will *express* the cytokine at the primary tumor site, thereby closely mimicking cytokine release in *vivo* and eventually targeting the antitumor response with minimal side effects. Animal models have shown that the local production of various cytokines by direct *injection* or by gene therapy can induce a strong antitumor response that results in long-lived immunity and, occasionally, in the abrogation of established tumors. A table is provided that lists studies examining the effects of some candidate cytokines (e.g. interleukin-2, interleukin-4, interleukin-7, interferon-gamma, *tumor* *necrosis* *factor* , granulocyte-macrophage colony stimulating factor and somatomedin-C antisense) in humans for treatment of e.g. melanoma, advanced cancer, brain tumor, colon cancer, lung cancer, neuroblastoma, renal cell carcinoma, and lymphoma, using e.g. retro virus *transfection*, lipofection or electroporation. Genetic modification of lymphocytes, endothelial cells and fibroblasts for cytokine delivery is discussed. (49 ref)
DESCRIPTORS: cancer cytokine-mediated gene therapy, review tumor (Vol.14, No.15)
SECTION: PHARMACEUTICALS-Clinical Genetic Techniques; GENETIC ENGINEERING AND FERMENTATION-Nucleic Acid Technology (D7,A1)

11/9/236 (Item 9 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs


163178 DBA Accession No.: 94-05729 PATENT
Tumor gene therapy using DNA encoding cytokine - e.g. interleukin-2, -4, -7, macrophage colony stimulating factor, interferon-gamma or *tumor* *necrosis* *factor*
PATENT ASSIGNEE: Imperial-Cancer-Res.Technol. 1994
PATENT NUMBER: WO 9404196 PATENT DATE: 940303 WPI ACCESSION NO.: 94-082848 (9410)
PRIORITY APPLIC. NO.: GB 934024 APPLIC. DATE: 930227
NATIONAL APPLIC. NO.: WO 93GB1730 APPLIC. DATE: 930816
LANGUAGE: English
ABSTRACT: A novel DNA construct (I) comprises a means for *expressing* a sequence encoding a cytokine in a tumor cell, and optionally a B7 coding sequence, a means for its *expression* in a tumor cell and a means for selectively delivering (I) to a tumor. Also claimed is a method of treating a tumor and/or ameliorating metastasis by delivery of (I) into tumor cells, where (I) *expresses* at least 2 cytokines in the tumor cells. The tumor cells are especially melanoma, mamma

carcinoma, colon carcinoma, pancreas carcinoma and prostate carcinoma cells, and the cytokine is interleukin (IL)-2, IL-4, macrophage colony stimulating factor (M-CSF), interferon-gamma, *tumor* *necrosis* *factor* or interleukin-7. (I) preferably contains coding sequences for IL-2, IL-4 and M-CSF in a 1:1:1 molar ratio, and gene *expression* may be under the control of the c-erb-B2 or c-erb-B3 gene promoter, the CEA promoter, MUC1 gene promoter or the PSA gene promoter. Naked DNA may be *injected* directly into the tumor, or selective delivery may be applied using liposomes (lipofection) carrying tumor cell targeting means, or a retro virus or adeno virus vector specific for the tumor cells. (107pp)
DESCRIPTORS: DNA construct for cytokine e.g. interleukin-2, -4, -7, macrophage colony stimulating factor, interferon-gamma, *tumor* *necrosis* *factor* gene *expression* in *vivo*, pot. tumor gene therapy retro virus adeno virus vector lipofection lymphokine antitumor melanoma mamma colon pancreas prostate carcinoma (Vol.13, No.10)
SECTION: PHARMACEUTICALS-Clinical Genetic Techniques; GENETIC ENGINEERING AND FERMENTATION-Nucleic Acid Technology (D7,A1)
?t s20/9/8

20/9/8 (Item 8 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


13161817 BIOSIS Number: 99161817
Interdisciplinary operative therapy for renal *tumors* with intracardiac *tumor* thrombosis
Akcetin Z; Schafhauser W; Kuehn R; Scheele J; Weniger J; Schrott K M
Poliklinik Urol. Univ. Halle-Wittenberg, Magdeburger Strasse 16, D-06097 Halle, Germany
Urologe Ausgabe A 35 (2). 1996. 115-119.
Full Journal Title: Urologe Ausgabe A
ISSN: 0340-2592
Language: GERMAN
Print Number: Biological Abstracts Vol. 102 Iss. 007 Ref. 109948
A combination of increased perioperative morbidity, together with the technical difficulty of an R 0 (curative) resection, is responsible for the poor prognostic factors of supradiaphragmatically extending renal *tumors*. Six patients aged 53-70 years with vena cava thrombosis extending into the right atrium or ventricle underwent en bloc resection of the primary *tumor* and *tumor* thrombus removal. If the atrial *tumor* mass was large or extended into the ventricle, resection was performed during cardiopulmonary arrest using a cardiopulmonary bypass method with the patient in deep hypothermia ( lt 18 degree C). Alternatively, if the cardiac *tumor* infiltration was minimal, resection was performed during an optionally short cardiopulmonary arrest period using a cardiopulmonary bypass method with the patient in hypothermia (23 degree C). The operative procedure was determined by intracardiac *tumor* extension, *tumor* wall adhesions and *tumor* wall infiltrations, all of which were assessed intraoperatively by vena cava sonography. Six patients were strongly symptomatic preoperatively. Three developed sudden life-threatening cardiopulmonary insufficiency, possibly due to longer-lasting tricuspital valve prolapse with a consecutive right-to-left shunt through a newly reopened foramen ovale. One patient died 14 months postoperatively because of multiple metastases (hepatic, pulmonary and bone). One patient is still alive and has had a local recurrence for 2 months, which was diagnosed 65 months postoperatively. The remaining four patients are alive and well.

They have been *tumor*-free for extended periods of time (29, 34, 62 and 84 months, respectively).
Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; VENA CAVA THROMBOSIS; MULTIPLE METASTASES; RIGHT-TO-LEFT SHUNT
Concept Codes:
*11107 Anatomy and Histology, General and Comparative-Regeneration and Transplantation (1971- )
*12502 Pathology, General and Miscellaneous-General
*12504 Pathology, General and Miscellaneous-Diagnostic
*12512 Pathology, General and Miscellaneous-Therapy (1971- )
*14501 Cardiovascular System-General; Methods
*15001 Blood, Blood-Forming Organs and Body Fluids-General; Methods
*15501 Urinary System and External Secretions-General; Methods
*16001 Respiratory System-General; Methods
*23001 Temperature: Its Measurement, Effects and Regulation-General Measurement and Methods
*24002 Neoplasms and Neoplastic Agents-General
Biosystematic Codes:
86215 Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans
?t s38/9/1-4

38/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


10514071 BIOSIS Number: 96114071
USE OF TISSUE-SPECIFIC EXPRESSION OF THE HERPES SIMPLEX VIRUS THYMIDINE KINASE *GENE* TO INHIBIT GROWTH OF ESTABLISHED MURINE MELANOMAS FOLLOWING DIRECT *INTRATUMORAL* *INJECTION* OF DNA
VILE R G; HART I R
BIOL. METASTASIS LAB., IMPERIAL CANCER RES. FUND, 44 LINCOLN'S INN FIELDS, LONDON, WC2A 3PX, UK.
CANCER RES 53 (17). 1993. 3860-3864. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
We report here the use of the 5' flanking region of the murine tyrosinase *gene* to direct expression of the herpes simplex virus thymidine kinase (tk) *gene* specifically to murine melanoma cells, whilst not permitting expression in a range of other cell types. Expression of the herpes simplex virus tk *gene* from the tyrosinase promoter in melanoma cells rendered them sensitive to killing by ganciclovir (100% cell death of a tk-expressing B16 clone after 12 days in culture at 1.mu.g/mi ganciclovir). We also observed a substantial bystander killing effect when expressing cells were mixed with nontransfected parental B16 cells. When transfected murine melanoma cells expressing tk were injected into syngeneic mice both their tumorigenicity and experimental metastatic potential were abrogated completely when the mice were treated with ganciclovir (27 of 28 mice treated with water developed progressively growing tumors versus 1 of 30 in the ganciclovir-treated group). Direct injection of the tk *gene* under control of the tyrosinase promoter into established tumors in mice, followed by treatment with ganciclovir, led to significant reductions in resultant tumor size relative to the size of tumor developing in mice treated with water (median tumor weight, 1.65 g versus 2.75 g). Therefore, direct transfer of recombinant genes by injection of DNA can significantly reduce established tumor burden in vivo.

Descriptors/Keywords: GANCICLOVIR ANTIVIRAL-DRUG TUMOR BURDEN
TUMORIGENICITY METASTATIC POTENTIAL
Concept Codes:
*15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*18506 Integumentary System-Pathology
*24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects
*24007 Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis
*31500 Genetics of Bacteria and Viruses
*36006 Medical and Clinical Microbiology-Virology
*38506 Chemotherapy-Antiviral Agents
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
Biosystematic Codes:
02612 Herpesviridae (1993- )
86375 Muridae
Super Taxa:
Microorganisms; Viruses; Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Rodents

38/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


10490141 BIOSIS Number: 96090141
REGRESSION OF ESTABLISHED MACROSCOPIC LIVER METASTASES AFTER IN-SITU TRANSDUCTION OF A SUICIDE *GENE*
CARUSO M; PANIS Y; GAGANDEEP S; HOUSSIN D; SALZMANN J-L; KLATZMANN D
LAB. BIOL. GENET. PATHOL. IMMUNITARIES, CENT. NATL. RECHERCHE SCIENTIFIQUE, UNITE RECHERCHE ASSOCIEE 1463, HOPITTAL PITIE-SALPETRIERE, 83 BOULEVARD L'HOPITAL, 75651 PARIS CEDEX 13, FR.
PROC NATL ACAD SCI U S A 90 (15). 1993. 7024-7028. CODEN: PNASA
Full Journal Title: Proceedings of the National Academy of Sciences of the United States of America
Language: ENGLISH
The herpes simplex virus type 1 thymidine kinase (HSV1-TK) converts nontoxic nucleoside analogs such as ganciclovir into phosphorylated compounds that act as chain terminators and specifically kill dividing cells. This property could be exploited for the treatment of tumors that are made up of rapidly dividing cells invading a nonproliferating tissue. For this purpose, specific expression of the suicide *gene* into dividing tumor cells can be further targeted by using retroviral-mediated *gene* transfer. We investigated whether the direct intratumoral transduction of a suicide *gene* might induce the elimination of malignant solid tumors. Rats with established macroscopic liver metastases were given an *intratumoral* *injection* of packaging cells producing either HSV1-TK- or lacZ-expressing recombinant retroviral particles. All rats were next treated with ganciclovir. A dramatic regression of the tumor volume was observed in the HSV1-TK-treated animals. The residual tumors were mostly made up of a massive fibrotic reaction, with the mean cancer cell mass being reduced .apprxeq.60-fold compared to controls. In some animals, the residual tumors were devoid of cancer cells. This treatment efficacy appears in part due to a "bystander effect" in which phosphorylated ganciclovir could be transferred from cell to cell and to an active local immune reaction evidenced by massive infiltration of the tumors by macrophages and both CD4+ and CD8+ lymphocytes. This efficient therapeutic approach might be an ultimate treatment for disseminated liver metastases in humans and could

also be applied to treatment of a large variety of solid tumors.
Descriptors/Keywords: RAT HERPES SIMPLEX VIRUS TYPE 1 THYMIDINE KINASE *GENE* THERAPY RETROVIRAL *VECTOR* NUCLEOSIDE ANALOGS CANCER TUMORAL MACROPHAGE INFILTRATION HISTOPATHOLOGY
Concept Codes:
*02506 Cytology and Cytochemistry-Animal
*03506 Genetics and Cytogenetics-Animal
*10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines
*10804 Enzymes-Methods
*12512 Pathology, General and Miscellaneous-Therapy (1971- )
*14002 Digestive System-Anatomy
*14006 Digestive System-Pathology
*15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*24003 Neoplasms and Neoplastic Agents-Immunology
*24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects
*24008 Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy
*31500 Genetics of Bacteria and Viruses
*33506 Virology-Animal Host Viruses
*34508 Immunology and Immunochemistry-Immunopathology, Tissue Immunology
01056 Microscopy Techniques-Histology and Histochemistry
10052 Biochemical Methods-Nucleic Acids, Purines and Pyrimidines
22005 Pharmacology-Clinical Pharmacology (1972- )
Biosystematic Codes:
02612 Herpesviridae (1993- )
86375 Muridae
Super Taxa:
Microorganisms; Viruses; Animals; Chordates; Vertebrates; Nonhuman Vertebrates; Mammals; Nonhuman Mammals; Rodents

38/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)


10052475 BIOSIS Number: 95052475
IN-SITU RETROVIRAL-MEDIATED *GENE* TRANSFER FOR THE TREATMENT OF BRAIN TUMORS IN RATS
RAM Z; CULVER K W; WALBRDIGE S; BLAESE R M; OLDFIELD E H
SURGICAL NEUROL. BRANCH, NATIONAL INST. NEUROL. DISORDERS STROKE CLINICAL CENTER, NIH, BUILDING 10, ROOM 5D37, 9000 ROCKVILLE PIKE, BETHESDA, MD. 20892.
CANCER RES 53 (1). 1993. 83-88. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
*Gene* transfer with vectors derived from murine retroviruses is restricted to cells which are proliferating and synthesizing DNA at the time of infection. This suggests that retroviral-mediated *gene* transfer might permit targeting of *gene* integration into malignant cells in organ composed mainly of quiescent nonproliferating cells, such as in the brain. Accordingly, selective introduction of genes encoding for susceptibility to otherwise nontoxic drugs ("suicide" genes) into proliferating brain tumors may be used to treat this cancer. We investigated the efficacy and dynamics of in vivo transduction of growing brain tumors with the herpes simplex-thymidine kinase *gene* followed by administration of the antiviral drug ganciclovir. Ganciclovir is phosphorylated by thymidine kinase to

toxic triphosphates that interfere with DNA synthesis, resulting in the preferential death of the transduced tumor cells. Rats inoculated with 4 .times. 104 9L gliosarcoma cells into the frontal lobe were treated 7 days later with an intratumoral stereotaxic injection of murine fibroblasts (NIH 3T3 cells) that were producing a retroviral *vector* containing the herpes simplex-thymidine kinase *gene*. Controls received *vector* producer and nonproducer NIH 3T3 cell lines containing the Escherichia coli lacZ (.beta.-galactosidase) *gene* as well as nonproducer NIH 3T3 cells containing the thymidine kinase *gene*. The animals were rested for 7 days to allow time for in situ transduction of the proliferating tumor cells with the herpes-thymidine kinase retroviral *vector*. The animals were then treated with ganciclovir, 15 mg/kg i.p. twice a day for 14 days. Gliomas receiving an injection of 3-5 .times. 106 thymidine kinase producer cells regressed completely in 23 of 30 rats given ganciclovir therapy, while 25 of 26 control rats developed large tumors. *Intratumoral* *injection* of a lower concentration of thymidine kinase *vector* producer cells (1.8 .times. 106) resulted in a lower frequency of tumor regression (5 of 13 rats). To estimate the efficiency in vivo *gene* transfer, 9L brain tumors were given injections of 5 .times. 106 .beta.-galactosidase *vector* producer cells. 5-Bromo-4-chloro-3-indolyl-.beta.-D-galactopyranoside staining revealed maximal staining of .beta.-galactosidase within the tumor 7-14 days after injection of the *vector* producer cells. In vivo transduction rates in harvested tumors ranged from 10 to 70%. There was no evidence of transduction of the surrounding normal neural tissue. Occasional blood vessel endothelial cells within or adjacent to the tumor were observed to be 5-bromo-4-chloro-3-indolyl-.beta.-D-galactopyranaside positive. It is probable that destruction of this local vasculature with ganciclovir therapy also contributes to the efficacy of tumor regression. Our results substantiate the feasibility of this approach for the treatment of malignant brain tumors in humans.

Descriptors/Keywords: HUMAN GLIOSARCOMA GANCICLOVIR ANTINEOPLASTIC-DRUG VASCULATURE DESTRUCTION

Concept Codes:

| | |
|---|---|
| *03506 | Genetics and Cytogenetics-Animal |
| *14508 | Cardiovascular System-Blood Vessel Pathology |
| *20506 | Nervous System-Pathology |
| *22010 | Pharmacology-Cardiovascular System |
| *22024 | Pharmacology-Neuropharmacology |
| *24008 | Neoplasms and Neoplastic Agents-Therapeutic Agents; Therapy |
| *31500 | Genetics of Bacteria and Viruses |
| *36006 | Medical and Clinical Microbiology-Virology |
| 10062 | Biochemical Studies-Nucleic Acids, Purines and Pyrimidines |
| 18006 | Bones, Joints, Fasciae, Connective and Adipose Tissue-Pathology |
| 22005 | Pharmacology-Clinical Pharmacology (1972- ) |
| 22012 | Pharmacology-Connective Tissue, Bone and Collagen-Acting Drugs |
| 33506 | Virology-Animal Host Viruses |
| 38506 | Chemotherapy-Antiviral Agents |

Biosystematic Codes:

| | |
|---|---|
| 02623 | Retroviridae (1993- ) |
| 86215 | Hominidae |
| 86375 | Muridae |

Super Taxa:

Microorganisms; Viruses; Animals; Chordates; Vertebrates; Mammals; Primates; Humans; Nonhuman Vertebrates; Nonhuman Mammals; Rodents

38/9/4 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

096929 DBA Accession No.: 89-14920
In vitro and in vivo expression of human interferon-beta in glioma cells transfected with its *gene* encapsulated in liposomes - potential *gene* therapy (conference abstract)
AUTHOR: Mizuno M; Yoshida J; Sugita K; Koshizaka T; Hayashi Y; Yagi K
CORPORATE SOURCE: Department of Neurosurgery, Nagoya University School of Medicine, Nagoya 466, Japan.
JOURNAL: J.Interferon Res. (9, Suppl.2, S151) 1989
CODEN: JIREDJ
LANGUAGE: English
ABSTRACT: As a preliminary study for *gene* therapy of patients with malignant glioma, liposomes encapsulating the human interferon-beta (IFN-beta) *gene* were targeted to glioma cells. A new transfection system using liposome positive charges on their surface and encapsulating plasmids containing the human IFN-beta *gene* (pSV2IFN-beta) was constructed. Glioma cells transfected in vitro with the liposomes produced human IFN-beta. A monoclonal antibody (MAb) specific for glioma-associated antigen was coupled to the liposomes and targeted to glioma cells. The production of human IFN-beta was increased 10-fold by MAb addition. An in vivo experiment for expression of human IFN-beta was performed using transplants of human glioma cells and nude mice. The glioma cells continuously secreted high levels (over 100 U/ml) of human IFN-beta into the cystic fluid of the tumor following *intratumoral* *injection* of *vector* plasmid pSV2IFN-beta-encapsulating liposomes. (0 ref)
DESCRIPTORS: human recombinant interferon-beta prep., *gene* cloning, liposome-mediated *vector* plasmid pSV2IFN-beta expression in glioma cell culture, pot. tumor *gene* therapy, lipofection mammal protein *gene* transmission transformation
SECTION: Pharmaceuticals-Interferon; Microbiology-Genetics; Cell Culture-Animal Cell Culture (D3,A1,J1)
?t s42/9/206

42/9/206 (Item 16 from file: 154)
DIALOG(R)File 154:MEDLINE(R)


08135784 92273784
Cytokine *gene* transfer in *tumor* cells as an approach to antitumor therapy.
Colombo MP; Mattei S; Parmiani G
Division of Experimental Oncology D, Istituto Nazionale per lo Studio e la Cura dei Tumori, Milan, Italy.
Int J Clin Lab Res (GERMANY) 1992, 21 (4) p278-82, ISSN 0940-5437
Journal Code: A81
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9209
Subfile: INDEX MEDICUS
The transfer of cytokine genes into cancer cells, resulting in cytokine release directly at the site of *tumor* growth, has proven effective in inhibiting *tumor* growth in the absence of any toxic effect. Some cytokines induce *tumor* suppression even in T-cell-deficient mice, suggesting their potential therapeutic effect in poorly immunogenic *tumors* ; other cytokines induce memory T cells that protect mice from

subsequent *tumor* *injection*. The effects of cytokine genes transferred into *tumor* cells are summarized and implications discussed. (30 Refs.)

Tags: Animal; Human

Descriptors: *Cytokines--Genetics--GE; *Neoplasms--Therapy--TH; *Transfection; Cytokines--Adverse Effects--AE; Cytokines--Therapeutic Use--TU; Cytotoxicity, Immunologic; Mice; Mice, *Nude*; Neoplasm Transplantation; Neoplasms--Immunology--IM; Neoplasms, Experimental--Immunology--IM; Neoplasms, Experimental--Therapy--TH; Recombinant Proteins--Genetics--GE; Recombinant Proteins--Therapeutic Use--TU; *Tumor* Cells, Cultured--Metabolism--ME; *Tumor* Cells, Cultured--Transplantation--TR

CAS Registry No.: 0 (Cytokines); 0 (Recombinant Proteins)